PB 188 080

AIR POLLUTION ASPECTS OF ASBESTOS

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Litton Systems, Incorporated
Bethesda, Maryland

September 1969

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AIR POLLUTION ASPECTS
OF
ASBESTOS

Prepared for the
National Air Pollution Control Administration
Consumer Protection & Environmental Health Service
Department of Health, Education, and Welfare
(Contract No. PH-22-68-25)

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September 1969

FOREWORD

As the concern for air quality grows, so does the concern over the less ubiquitous but potentially harmful contaminants that are in our atmosphere. Thirty such pollutants have been identified, and available information has been summarized in a series of reports describing their sources, distribution, effects, and control technology for their abatement.

A total of 27 reports have been prepared covering the 30 pollutants. These reports were developed under contract for the National Air Pollution Control Administration (NAPCA) by Litton Systems, Inc. The complete listing is as follows:

- Aeroallergens (pollens)
- Aldehydes (includes acrolein and formaldehyde)
- Ammonia
- Arsenic and Its Compounds
- Asbestos
- Barium and Its Compounds
- Beryllium and Its Compounds
- Biological Aerosols (microorganisms)
- Boron and Its Compounds
- Cadmium and Its Compounds
- Chlorine Gas
- Chromium and Its Compounds (includes chromic acid)
- Ethylene
- Hydrochloric Acid
- Hydrogen Sulfide
- Iron and Its Compounds
- Manganese and Its Compounds
- Mercury and Its Compounds
- Nickel and Its Compounds
- Odorous Compounds
- Organic Carcinogens
- Pesticides
- Phosphorus and Its Compounds
- Radioactive Substances
- Selenium and Its Compounds
- Vanadium and Its Compounds
- Zinc and Its Compounds

These reports represent current state-of-the-art literature reviews supplemented by discussions with selected knowledgeable individuals both within and outside the Federal Government. They do not however presume to be a synthesis of available information but rather a summary without an attempt to interpret or reconcile conflicting data. The reports are necessarily limited in their discussion of health effects for some pollutants to descriptions of occupational health exposures and animal laboratory studies since only a few epidemiologic studies were available.

Initially these reports were generally intended as internal documents within NAPCA to provide a basis for sound decision-making on program guidance for future research activities and to allow ranking of future activities relating to the development of criteria and control technology documents. However, it is apparent that these reports may also be of significant value to many others in air pollution control, such as State or local air pollution control officials, as a library of information on which to base informed decisions on pollutants to be controlled in their geographic areas. Additionally, these reports may stimulate scientific investigators to pursue research in needed areas. They also provide for the interested citizen readily available information about a given pollutant. Therefore, they are being given wide distribution with the assumption that they will be used with full knowledge of their value and limitations.

This series of reports was compiled and prepared by the Litton personnel listed below:

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Appreciation is expressed to the many individuals both outside and within NAPCA who provided information and reviewed draft copies of these reports. Appreciation is also expressed to the NAPCA Office of Technical Information and Publications for their support in providing a significant portion of the technical literature.

ABSTRACT

Inhalation of asbestos may cause asbestosis, pleural or peritoneal mesothelioma, or lung cancer. Mesothelioma is a rare form of cancer which occurs frequently in asbestos workers. All three of these diseases are fatal once they become established. The dose necessary to produce asbestosis has been estimated to be 50 to 60 million particles per cubic foot-years. No information is available on the dose necessary to induce cancer. Random autopsies of lungs have shown "asbestos bodies" in the lungs of one-fourth to one-half of samples from urban populations. Thus, the apparent air pollution by asbestos reaches a large number of people.

Animals have been shown to develop asbestosis and cancer after exposure to asbestos.

No information has been found on the effects of asbestos air pollution on plants or materials.

The likely sources of asbestos air pollution are uses of the asbestos products in the construction industry and asbestos mines and factories. Observations in Finland and Russia indicate that asbestos does pollute air near mines and factories. However, no measurements were reported of the concentration of asbestos near likely sources in the United States. A concentration in urban air of 600 to 6,000 particles per cubic meter has been estimated.

Bag filters have been used in factories to control

asbestos emissions; the cost of this type of control in a British factory was approximately 27.5 percent of the total capital cost and about 7 percent of the operating cost. No information has been found on the costs of damage resulting from asbestos air pollution.

No satisfactory analytical method is available to determine asbestos in the atmosphere.

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1. INTRODUCTION

Asbestos is a general name given to a variety of useful fibrous minerals. The value of asbestos ensues from the indestructible nature of products fabricated from the various grades of mineral fibers. The major asbestos minerals are chrysotile, crocidolite, amosite, and anthophyllite, while tremolite and actinolite are considerably less important. Over 90 percent of the asbestos is chrysotile. The United States uses about one-fourth of the world production of this substance, practically all imported from Canada and Africa.

Inhalation of asbestos dust has long been recognized as an industrial hazard.[161,165] Early in this century, exposure to high concentrations of the fibrous dust was causally associated with asbestosis. In 1935, evidence began to accumulate that cancer of the lung is also associated with inhalation of asbestos.[55] More recently, certain rare cancers, pleural mesotheliomas and peritoneal mesotheliomas, have been associated with inhalation of asbestos fibers by asbestos workers.[176] Heimann[92] states that "The finding of several such rare tumors in any given group makes that group suspect of having special and distinct environmental characteristics, in this case, exposure to asbestos dust."

Nonoccupational environmental exposure to asbestos was found as early as 1927 when Haddow[81] reported finding so-called "asbestos bodies" in the lungs of a person living near an asbestos factory. Since then, several investigators[121,175,186,202,226] have reported finding neighborhood cases. The subject of nonoccupational environmental exposure to asbestos assumed a new dimension beginning with the reports of Thomson and his colleagues.[213-216] After examining the lungs in consecutive autopsies, they found that approximately one-fourth of the populations in both Capetown, South Africa, and Miami, Fla., have "asbestos bodies" in their lungs. Other investigators have confirmed that one-fourth to one-half of the population in Pittsburgh,[42] San Francisco,[43] Milan,[67] Glasgow,[188] New York,[195] Montreal,[7] Jerusalem,[184] Finland,[167] and Sweden[82] have "asbestos bodies" in their lungs. These findings indicate that either these asbestos particles or other particles that resemble asbestos in many ways, including the way in which the body reacts to them, are being inhaled either with the ambient urban air or through direct exposure to asbestos.

2. EFFECTS

2.1 Effects on Humans

Asbestosis (a diffuse pulmonary fibrosis), pleural calcification, pleural plaques, lung cancer, and pleural and peritoneal mesotheliomas can result from exposure to asbestos. Asbestos bodies are commonly found in the lungs of persons exhibiting these complications. Diagnosis of any of these or finding "asbestos bodies" in the lungs signifies the need to review the case history for previous asbestos exposure. Surveys of people living or working near asbestos mines and factories have revealed that many nonoccupational cases of asbestosis and mesothelioma have occurred either from the polluted air or from asbestos carried home on the workers' clothing.[175,176,178] However, in many cases no exposure to asbestos can be established.

The fate of the asbestos fiber once it is inhaled* and deposited in the lung is still questionable. The short fibers, <0.5 μ in length, have been pathologically ignored, probably because they are much too narrow to be visible under a light microscope. The longer fibers which are encrusted in an iron-bearing protein (asbestos bodies) become easily visible. Wagner and Skidmore[228] and Morris et al.[172] have shown that rats which have inhaled asbestos lose the asbestos (probably the short fibers) from their lungs. The biological half-life for asbestos appears to be 20 to 90 days, depending on the mineral type. Some of the fibers are removed by phagocytosis to the lymph nodes.

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*A discussion on respirable fibers is presented in Appendix B (page 92).

2.1.1 Asbestosis

Among asbestos workers, evidence of pulmonary asbestosis is common. This condition results in a diffuse fibrosis, usually in the lower lobes of the lung. Pulmonary asbestosis has been called a monosymptomatic disease, with dyspnea as the main complaint.[197] The British Occupational Hygiene Society[136] has reported that basal rales are the first symptoms of asbestosis.

Asbestosis usually develops after long exposure to high concentrations of asbestos dust. The risk varies directly with the length of exposure and the dust concentration. Following continued exposure to high concentrations of dust, asbestosis may develop fully in 7 to 9 years and may cause death as early as 13 years from onset of exposure. The common exposure period before recognition of asbestosis (as observed among asbestos workers) is 20 to 40 years, with death following about 2 to 10 years later. Once established, asbestosis progresses even after the exposure to dust ceases: illness or death can occur long after exposure to concentrations not producing immediate effects.[197]

The prolonged latency period between exposure and the first signs of asbestosis makes it difficult to establish dose-time relationships. Cooper[43] suggested that a time-weighted average concentration of asbestos fibers of 5 mppcf* is too high. He cites Wells'[234] idea that multiplying average counts by years of exposure provides a rough guide to the total dose allowable. After 50 to 60-mppcf-years, workers began to show evidence of asbestosis. At an average concentration of 5 mppcf, this total allowable dose would be reached in 10 to 12 years. Unfortunately, dust concentrations have been infrequently reported and measurements have been hampered by the varied nature of the sources. Marr[153] and Selikoff _et_ _al_.[191] have reported that insulation workers are exposed to dust concentrations below 5 mppcf, yet have exhibited a high prevalence of asbestosis. Thus, Cooper's argument is strengthened.

In 1946, there were about 700 cases of asbestosis in Germany among a total of approximately 8,000 employees in the asbestos industry.[108] Wegelius[235] found 125 cases of asbestosis of the lung in X-ray examinations of 476 asbestos workers in one company in Finland. Of 132 asbestos workers examined by Bohme,[26] 29 percent showed X-ray evidence of asbestosis. The occurrence of asbestosis in members of this worker group rose with the duration of the employment: 5 percent in workers exposed to asbestos for less than 3 years; 56 percent for those employed for 5 to 10 years, and 79 percent for those with over 10 years' exposure. A similar morbidity of 80 percent among

---

*5 million particles per cubic foot based on total dust count and 8-hour-day, 40-hour-week exposure.

English asbestos workers with over 20 years of employment was reported by Merewether and Price[165] in 1930. Noro[177] noted that the incidence of asbestosis was 65 percent in 167 asbestos workers studied by X-ray. Selikoff _et al._[197] investigated 1,522 asbestos insulation workers in the New York-New Jersey metropolitan area. Among 392 individuals examined more than 20 years from the onset of exposure, X-ray evidence of asbestosis was found in 339. In half of these, the asbestosis was moderate or extensive. In individuals with less than 20 years of exposure, radiological evidence of asbestosis was less frequent and when present, was much less likely to be extensive.

2.1.2 Pleural Calcification and Plaques

Pleural calcification resulting from exposure to asbestos is usually bilateral and affects the parietal pleura.[120] Selikoff[193] stated that bilateral pleural calcification involving the diaphragm is diagnostic of asbestosis. This pleural calcification can be readily identified by X-ray.[117]

Kiviluoto[120] discovered 499 cases of pleural calcification during a community X-ray survey of 6,312 adults in the Kuusjari commune in Finland. In Ilomantsi commune he found no such calcification among 7,101 adults. He observed that the Kuusjari commune contained an asbestos mine and suggested that these people had been subject to a localized environmental asbestos exposure. This investigator[121] also observed 77 cases of pleural plaques out of 35,000 routine chest X-rays. The case histories of these 77 revealed that 52 had previous exposure to asbestos either in the home or in their occupations, where they handled asbestos products. Of the other 25 persons, 16 were questioned and no previous asbestos exposure could be ascertained. Raunio[186] continued the study and found 1,516 adult cases of pleural calcification from 633,201 X-rays taken in 13 Finnish towns and 106 rural communes. In Tuusniemi commune, where an asbestos quarry is located, pleural calcification was found in 9 percent of the population; in urban populations it was found in 0.7 percent of the people; and in rural areas calcifications were found in only 7 out of 265,273 people examined (0.002 percent). However, Meurman[167] found the pleural plaques were common (39.3 percent of his cases) in all Finland.

Approximately 2.8 percent of the agricultural workers in Czechoslovakia[105] and Bulgaria[238] have also been shown to have appreciable pleural calcification. In Bulgaria the soils worked by the farmers contained asbestos; even stone fences were made of outcrops of anthophyllite mineral. However, in Czechoslovakia no asbestos or known exposure to asbestos was found. After examining children living in the city of Asbest in the Soviet Union, Bobyleva et al.[24,25] concluded that they were suffering from impaired health caused by air pollution from asbestos plants.

2.1.3 Cancer

2.1.3.1 Cancer of the Lung

The most common complication of asbestosis is cancer of the lung. However, cancer of the lung apparently induced by asbestos may appear unaccompanied by asbestosis.

The association of lung cancer with exposure to asbestos dust has been the subject of many investigations in the second quarter of this century. In 1935, Lynch and Smith[149] in the United States described lung cancer found during autopsy of a patient with asbestosis. According to Homburger's data,[101] over a 20-year period eight cases of asbestosis were found in 4,137 autopsies at the medical school of Yale University. Of these eight asbestosis cases, four were associated with lung cancer (50 percent). In contrast, lung cancer was found in only two (12 percent) of 17 cases of silicosis.

Lynch and Cannon[147] in 1949 found lung cancer in only three cases (7.5 percent) of 40 patients with asbestosis. Gloyne[71] in 1951 reported that according to autopsy data for the London Hospital of Chest Diseases, lung cancer was observed in 14 percent of the 121 patients with asbestosis, but in only 6.9 percent of those with silicosis.

Usually, the lung cancer incidence in men is four to six times higher than in women, but among patients with asbestosis it is only twice as high for men. This has been confirmed by Bohlig, Jacob, and Kalliabis.[29] They note that among women working in the asbestos industry, lung cancer is observed at an earlier age than among the rest of the population. In his review, Behrens[19] reported 44 cases of lung cancer (14 percent) in 309 autopsies of patients with asbestosis. After examining the data of various authors, Isselbacher et al.[111] reported that of 603 persons with asbestosis, lung cancer was found in 83 (13.8 percent) at autopsy. Hueper[108] and Doll[55] record an even higher percentage (15 percent). Doll emphasizes that the majority of those who died had worked under conditions of high dust content in the air.

Braun and Truan[33] found lung cancer in 12 (three cases were not conclusive) of 187 workers in the Canadian asbestos industry who had died. Boehme[26] reported 74 patients with asbestosis; lung cancer was found in six of these. The average age of the patients was 53; the average period from the beginning of work to death was 28 years. Keal[114] points out that of 30 patients with asbestosis, 14 (47 percent) died of lung cancer and 10 from carcinoma of the peritoneum or ovaries.

Some additional statistics on the association of lung cancer with asbestos workers are listed in Table 5 in the Appendix.

Cancer of the lung produced by asbestos needs further study. The latent period between exposure and evidence of carcinoma may be even longer than for asbestosis. Nothing is known about the dose-time relationship. Cases of lung cancer have been observed when only a very short exposure or no exposure to asbestos was known. Furthermore, the low number of "asbestos bodies" observed in one-fourth to one-half of the urban population may be sufficient to cause cancer. Because the long "asbestos bodies" remain in the lungs, a person who has inhaled asbestos may carry the potential (for the rest of his life) to develop carcinoma of the lung. Moreover, it has not been determined whether more than one fiber is necessary to induce a malignant tumor. Cox[42] has suggested that the probability of cancer induction is proportional to the number of asbestos fibers, number of susceptible cells, the concentration of carcinogens on the fibers, and the time from exposure.

Why asbestos is carcinogenic is not clearly understood. At least three hypotheses have been advanced:

(1) That the fibers act as a physical irritant which after 20 to 30 years of constant irritation induces a tumor.

(2) That the fibers contain small amounts of carcinogens--such as benzo(a)pyrene, nickel, and chromium--which are eluted from the fibers by the serum in the lungs. These carcinogens then produce the cancer. Harington and Roe[88] have shown that (a) chrysotile contains little or no benzo(a)pyrene, but about 100 μg of chromium per g of fiber and 5,000 μg of nickel per g of fiber; (b) crocidolite contains 0.2 to 24 μg of benzo(a)pyrene per 100 g of fiber and negligible amounts of nickel and chromium; and (c) amosite contains 0.2 to 2.4 μg of benzo(a)pyrene per 100 g of fiber, 100 μg of nickel per g of fiber and 15 μg of chromium per g of fiber (see Appendix B, page 92). The authors have not only shown that these agents can be dissolved in the lung serum, but also that airborne fibers can adsorb

carcinogens present in the air. In studies of the adsorption of benzo(a)pyrene by asbestos, chrysotile adsorbed 100 percent of benzo(a)pyrene from solution after 48 hours at 37°C, compared with 40 percent for crocidolite and 10 percent for amosite. Harington and Roe suggest that these carcinogens can be adsorbed on airborne fibers. Thus, the fibers become a transporting vehicle to carry a concentrated quantity of carcinogens into the lung.

(3) That the fibers accumulate in the lung and are immobilized as "asbestos bodies" which disintegrate after 20 to 40 years. The resulting free particles cause asbestosis or carcinoma of the lung.

In addition, Hammond[83] has suggested that asbestos is a cocarcinogen; i.e., it increases the cancer-producing potential of small amounts of some other agent. In a study of asbestos workers, he and his colleagues[199] found cancer of the lung only in cigarette smokers. The number of deaths attributed to cancer of the lung was eight times higher in asbestos workers who smoked cigarettes than in smokers who were not exposed to asbestos (see Section 2.1.3.4).

2.1.3.2 Mesothelioma of the Pleura and Peritoneum

Primary tumors of the pleura and peritoneum are so rare that for years they were considered to be pathologic curiosities. In 1960 the first large series of cases of diffuse mesothelioma were reported by Wagner *et al.*[229] in South Africa.

In trying to explain this mysterious epidemic, Wagner,[226] noting that "asbestos bodies" were found in the lungs of some of their patients, obtained detailed life histories of these patients. By 1960, he was able to establish an association with exposure to the Cape of Good Hope asbestos fields, or the industrial use of asbestos, in 32 of 33 patients with histologically proved pleural mesothelioma. The majority of these patients had not actually worked with asbestos but had lived in the vicinity of the mines and mills, and some had left these areas of exposure as young children. The average period between exposure and development of the tumor was 20 to 40 years. By 1962, Wagner[226] had diagnosed a total of 87 pleural and two peritoneal mesotheliomas. In only two cases was it impossible to establish a history of exposure to asbestos dust. Of these 87 cases, 12 had been industrially exposed and the remainder had been environmentally exposed from living in the vicinity of the mills and dumps. This association between mesothelioma and asbestosis became even more intriguing when in 1955, Bonser *et al*.[30] described a series of 72 autopsies on patients with asbestosis in which four cases of peritoneal mesothelioma were found. Subsequently, Mancuso and Coulter[152] found five peritoneal mesotheliomas in 1,495 asbestos workers, and Hourihane,[102] upon reviewing the necropsy files of the London Hospital from 1917 to 1962, found 34 cases of mesothelioma, half of the pleura and the other half of the peritoneum. All of these patients had pulmonary asbestosis, even though in a few there was no history of exposure to the asbestos dust. In a subsequent study[103] Hourihane found 74 cases of mesothelioma in a London hospital over a 10-year period.

Borow *et al.*[31] report that in two years they observed 11 cases of mesothelioma during surgery in New Jersey. These cases, added to six others previously diagnosed by them, totaled 17 cases of mesothelioma, eight of which were peritoneal and nine pleural. They suggest that the high prevalence of these rare tumors in New Jersey can be explained by its close proximity to a major asbestos mill, where a large percentage of all the asbestos fiber mined in North America is converted to commercial use.

In an attempt to determine whether mesothelioma of the serosal surfaces was related in any way to asbestos exposure in the United States, Selikoff *et al.*[198] studied 307 consecutive deaths among asbestos insulation workers in the Northeastern United States. They found 10 deaths caused by four pleural and six peritoneal mesotheliomas. In addition, these workers had a high death rate attributed to cancer of the stomach, colon, and rectum. Of the 307 deaths, 40.4 percent were attributed to cancer, 5.5 percent to asbestosis, and 54.1 percent to other causes. In a second study, the investigators reviewed 26 consecutive autopsies of patients with asbestosis, and found four mesotheliomas of the pleura and three of the peritoneum.

A series of 83 patients from the London Hospital with a diagnosis of mesothelioma (confirmed by necropsy or biopsy) were studied by Newhouse and Thompson[176] for possible exposure to asbestos. The series consisted of 41 men and 42 women; 27 of the patients had peritoneal and 56 pleural tumors. Although the earliest death recorded from this group was in 1917, only 10 patients died before 1950, while 40 (48 percent) died between 1960 and 1964. In 76 of the 83 cases, full occupational and residential histories were obtained. Forty patients (52.6 percent) had a history of occupational exposure to asbestos or of domestic exposure (living in the house with an asbestos worker). In comparison, only 11.8 percent (9 of 76) of the patients from the same hospital suffering from other diseases had previous exposure to asbestos. There was also evidence that neighborhood exposures may be important. Among those in this study with no history of occupational or domestic exposures to asbestos, 30.6 percent of the mesothelioma patients and 7.6 percent of the inpatients with other diseases lived within half a mile of an asbestos factory. Of the 31 patients with occupational exposures to asbestos, only 10 held jobs scheduled under the British Asbestos Regulations of 1931. The interval between first exposure and the development of the terminal illness from mesothelioma ranged between 16 and 55 years. The duration of exposure varied widely, ranging from two months to over 50 years. In 47 patients in this mesothelioma series, lung tissue or sputum was available for examination. In 30 (62.5 percent), either asbestosis or the presence of asbestos bodies was noted.

Mesothelioma is now considered a frequent cause of death among asbestos workers. No attempt has been made to summarize the reports of mesothelioma, since they appear almost weekly in the current literature. So far, however, there appear to be few cases among the general population. Selikoff[198] reviewed 31,652 deaths among the general population of over 1,048,183 in the United States and found only three cases of mesothelioma. Moreover, he[195] points out that asbestos is not the only cause of mesothelioma; it has also been produced by silica[224] and polyurethane.[107]

2.1.3.3 Other Cancers

Extrapulmonary cancer has also been noted as a cause of death among asbestos workers. Kogan et al.[132] in 1966 reported 14 cases (31.1 percent), 11 women and 3 men. Four of the women died of uterine cancer, two of intestinal cancer, two of breast cancer, and one of liver cancer. Among the men, one died of stomach cancer, another of cancer of the urinary bladder, and a third of cancer of the prostate. Other cases of extrapulmonary cancer have been cited in Section 2.1.3.2.

2.1.3.4 Synergism

While the exact cause of lung cancer or pleural peritoneal mesothelioma induced by asbestos is not known, air pollution by other pollutants may accelerate the morbidity. One form of air pollution which is easily studied in individuals is smoking. Selikoff _et_ _al_.[199] recently studied the mortality of 370 asbestos insulation workers. In this group 24 men died of lung cancer and all had a history of smoking. (See Table 1 below.) This rate was eight times greater than the expected mortality rate, with age and smoking habits taken into account.

TABLE 1

DEATHS OF ASBESTOS INSULATION
WORKERS IN NEW YORK, 1963-67[199]
(By Smoking Habits)

Smoking Habits	No. of Cases[a]	Observed Deaths	Expected Deaths
Never smoked regularly	48	0	0.05
History of pipe, cigar-smoking only	39	0	0.13
History of regular cigarette smoking[b]	283	24	2.98
Total	370	24	3.16

[a] All with more than 20 years from onset of exposure.

[b] Includes cigarette smokers who also smoked pipes or cigars.

The blue asbestos, crocidolite, from South Africa is believed by many[53,79,136,140,209] to be much more carcinogenic than the other minerals of asbestos. Studies in Finland[167] indicate that anthophyllite also produces cancer. Wagner,[226] Smith et al.,[206] and recently Godwin and Jagatic,[73] reported that they had induced mesothelioma in mice using chrysotile. Moreover, animal experiments[76,78,98-100,113,172,225,228] have demonstrated that pulmonary complications occur with crocidolite, chrysotile, amosite, and anthophyllite. Some investigators believe that the fiber is not the carcinogenic agent, but rather the vehicle on which the carcinogens are carried to the target tissue. As stated earlier, chrysotile contains the most nickel and chromium of all asbestos minerals, while crocidolite contains the most benzo(a)pyrene.[87-89,159] All three of these impurities in asbestos are suspected of being carcinogenic. This knowledge, together with results which indicate carcinogens can be adsorbed from urban air or tobacco smoke, indicate that there may be no necessity to distinguish urban asbestos dust by mineral types.

2.1.4 "Asbestos Bodies"

As stated earlier, the recent finding of "asbestos bodies" in one-fourth to one-half of the urban population (see Table 6 in the Appendix) has added new impetus to the examination of asbestos as a general air pollutant.

An "asbestos body" has been defined as "an elongated golden or reddish-brown structure usually with clubbed ends; the shaft, which often shows a segmented or beaded appearance, is usually straight, but sometimes curvilinear with a tendency toward symmetry; usually it is from 3 to 5 μ in diameter and

20 to 100 µ in length. The coating contains iron demonstrable by Perle's stain (Prussian blue reaction), and probably composed of ferritin or ferritin-like material; it may cover the structure completely, masking the central fiber from direct view, or may be incomplete in the central portion of the shaft or in the interstices of the body, revealing an expanse of naked fiber.[220]

There is no doubt that the "asbestos bodies" formed in the lungs of the asbestos workers contain asbestos. Stumphius and Meyer[210] have investigated the composition of the "kernel" in the "asbestos bodies" removed from deceased shipyard workers (an occupational group with only indirect exposure). They found by electron microscopy and X-ray microanalyses that the "asbestos bodies" did indeed contain some minerals of asbestos. Out of 27 fibers, 17 were classified as serpentine (possibly chrysotile) and 10 as amphibole (possibly crocidolite). But what about the so-called "asbestos bodies" in the lungs of the general population? While these "asbestos bodies" probably contain some asbestos, there is no experimental evidence to date which shows what fractions contain asbestos or whether they contain any asbestos at all.[79,194] This subject is currently being debated by several investigators.[53,79,209] In fact, some object to the use of the term "asbestos bodies" and prefer to call them "ferruginous bodies." Moreover, Gross _et_ _al_.[79] have shown that "ferruginous bodies"—which appear identical under the microscope to those formed from asbestos—

can be produced from ceramic aluminium silicate, glass fibers, and silicon carbide fibers. However, Thomson[215] claims that a skilled pathologist can tell the difference. This controversy should soon be resolved, since both Gross[53] and Selikoff[194] are investigating the composition of the central fiber with the electron microprobe.

Generalized contamination with fibrous material is evidenced by the figures in Table 6 in the Appendix. Two other obvious conclusions are that "asbestos bodies" are found more frequently in older people than in younger, and more frequently in men than in women.

In none of the aforementioned studies has there been any quantitative count of "asbestos bodies" in the lungs of the general public. In most studies, "asbestos bodies" found were scanty, although in some instances the bodies were numerous.[214] In most of the investigations, the method of Thomson[214] was used (smears taken from basal lobes of the lung were examined and the asbestos bodies counted). Since only about one-half-millionth of a lung is examined, the finding of only one asbestos particle may be extrapolated to mean that perhaps a half-million fibers are present in the lung.[214] In one recent study in Pittsburgh, Utidjian et al.[220] made an attempt to quantify their results. In this study 98 percent of the 100 lungs examined contained "asbestos bodies" ("ferruginous bodies"). The results are given in Table 2. For comparison they suggested that if those cases with only one "asbestos body" were ignored, then

47 percent of the lungs examined contained two or more bodies, (20.5 percent of the women and 60.7 percent of the men). This 47 percent is in substantial agreement with the comparable 41 percent reported by Cauna[40] (see Table 6 in the Appendix) for residents of Pittsburgh.

TABLE 2

DISTRIBUTION OF "ASBESTOS (FERRUGINOUS) BODIES"
IN LUNGS IN PITTSBURGH[220]

Sex	No. of Cases	Mean Age	Fibers/Unit of Lung	Distribution (Percent)
Men	1	89	0	2
	21	62	1	37
	15	64	2-5	27
	19	70	>5	34
Total	56	65		100
Women	2	29	0	5
	29	57	1	66
	8	68	2-5	18
	5	64	>5	11
Total	44	60		100

2.2 Effects on Animals

2.2.1 Commercial and Domestic Animals

Kiviluoto[121] reported finding some asbestos bodies in a cow near an asbestos mine. In 1931, Shuster[201] reported finding extensive pulmonary fibrosis in a dog kept for ratting in an asbestos factory. The lungs of the dog also contained some asbestos fibers but no asbestos bodies were found.

Peacock and Peacock[182] have studied the effect of asbestos on white leghorn fowl. They tried dusting the birds with asbestos but found that the fibers did not penetrate far into the lung. When the fibers were injected into the lumen of the air sac, an immediate inflammatory reaction occurred, macrophages appeared and engulfed the fibers, and giant cell formation was observed. Four of the 17 chickens examined developed tumors: of the six injected with crocidolite, two developed tumors; of the 10 injected with amosite, only one developed a tumor; and one chicken injected with an unidentified variety of asbestos also developed a tumor.

2.2.2 Experimental Animals

Studies with experimental animals have shown that asbestos can induce fibrosis (asbestosis), cancer of the lung, and mesothelioma and can form "asbestos bodies."

Wagner[234] described experiments with rats in which 600 animals were exposed to various minerals of asbestos. The results are given in Table 3.

Gross and De Treville[76] made the following observations in studies on rats, hamsters, and guinea pigs. In rats that have inhaled high concentrations (86,000 $\mu g/m^3$) of chrysotile asbestos fibers for only a few months, minimal fibrotic lesions can be observed in the lungs of all animals. However, this form of asbestosis in rats is nonprogressive. In hamsters that have inhaled chrysotile dust, a fibrosis develops which is

TABLE 3

TYPE AND NUMBER OF TUMORS INDUCED BY INTRAPLEURAL INOCULATION OF S.P.F. RATS WITH ASBESTOS[234]

Asbestos Minerals	Animals Exposed	Animals Died	Misc. Nonmalig.	Misc. Tumors	Reticulum-cell Sarcomas	Mesotheliomas
Croc. I[a]	100	19	5	1	1	12
Croc. II[b]	100	10	4			6
Amosite	100	4	1		1	2
Chrysotile	100	25	5		2	18
Silica	100	11	1	1	2	7
Saline	100	5	2	1	2	
Total	600	74	18	3	8	45

[a] Crocidolite from Northwest Cape, South Africa.

[b] Crocidolite with oil extracted from it.

progressive; "asbestos bodies" are also formed. In guinea pigs, the inhalation of chrysotile dust produces fibrotic lesions similar to those observed in rats. The data indicate that the minimum time to produce asbestosis in rats and guinea pigs is 60 to 120 hours at an asbestos dust concentration of 86,000 $\mu g/m^3$. The investigators think the time required is shorter for hamsters.

Wagner and Skidmore[228] have shown that asbestos dust tends to accumulate in the alveoli arising directly from the respiratory bronchioles of rats. They also investigated the elimination of asbestos from the lungs as discussed in Section 2.1. Gross and De Treville[76] also observed a decrease in fiber content as the time from end of exposure increased.

Holt *et al.*[98] suggest from their observations on rats that fibrotic lesions in the lungs are caused by asbestos fibers (chrysotile) which are less than 3 μ long. Longer fibers are stored in the lungs as "asbestos bodies;" shorter fibers are removed from the lungs by phagocytosis. After some years, the larger fibers disintegrate, producing a large number of small particles. These small particles are then phagocytosed and produce fibrosis. They also suggest that asbestos is only fibrogenic when it is ingested by phagocytes.

Holt *et al.*[100] exposed guinea pigs to asbestos dust. After 14 days of exposure to dust, bronchiolitis was observed; after 21 days the damage was very severe, and "asbestos bodies" were observed along with asbestos fibers. After 226 days the lungs of animals dusted for more than 1,000 hours over a 78-day period developed a wide-spread, progressive fibrosis with only a few asbestos fibers and "asbestos bodies" present in the lung tissue. The experimenters concluded that asbestos fibers too small to be seen under the microscope will produce asbestosis.

In order to evaluate the possible distribution of asbestos within New York City, Selikoff[194,195] is now examining the lungs of rats found in the city for asbestos fibers.

In a similar investigation carried out in South Africa, wild animals captured near an asbestos mine were examined. However, the small number of "asbestos bodies" found in them precluded any conclusions.[234]

2.3 Effects on Plants

No information has been found in the literature on the effects of asbestos air pollution on plants.

2.4 Effects on Materials

No information has been found in the literature on the effects of asbestos air pollution on materials.

2.5 Environmental Standards

Both the American Conference of Governmental Industrial Hygienists[218] and the American Industrial Hygiene Association[110] have recommended an industrial threshold limit value for asbestos dust of 5,000,000 particles per cubic foot

(5 mppcf), based on total dust count and on an 8-hour day, 40-hour week. This value was recommended by Dreessen et al.[56] after a study of 541 employees in three textile plants using chrysotile. Only three doubtful cases of asbestosis were found in those exposed to dust concentrations of less than 5 mppcf, whereas numerous cases were found above 5 mppcf.

Recently, the British Occupational Hygiene Society[136] published its standards for chrysotile. The Society has recommended a maximum accumulated exposure of 2.8 mppcf-years (10^8 particle-years per m^3). For example, maximum doses of 0.056 mppcf-years (2×10^6 particle-years/m^3) for 50 years, 0.112 mppcf-years (4×10^6 particle-years/m^3) for 25 years, or 0.28 mppcf-years (10^7 particle-years/m^3) for 10 years are recommended. They have also recommended that dustiness be designated by categories according to the following scheme:

Dust Category	Concentration Averaged Over 3 Months (Million Particles/m^3)
Negligible	0-0.4
Low	.5-1.9
Medium	2.0-10
High	Over 10.0

Only fibers longer than 5.0 μ in length with a 3:1 length-to-breadth ratio are counted.

With these standards the risk of asbestosis may be reduced to 1 percent; that is, 1 percent of the workers exposed to a dose of 10^8 particle-years/m^3 would contract asbestosis.

3. SOURCES

3.1 Natural Occurrence

Asbestos is a broad term embracing several fibrous minerals. The minerals are divided into two groups: (1) Pyroxenes—chrysotile; (2) Amphiboles—crocidolite, amosite, tremolite, actinolite, and anthophyllite. Properties of these minerals are listed in Table 7 of the Appendix.

Asbestos probably occurs in nearly every country in the world, but only a few of the deposits are commercially valuable. Over 90 percent of the world asbestos production is chrysotile, and Canada is the major source of this mineral for the United States.[93] Table 8 in the Appendix lists the world production. From these figures an estimate of known free-world deposits is possible.

It is noteworthy that some soils near asbestos mines contain considerable quantities of asbestos. In Finland, farmers working these high-asbestos-content soils have been observed to suffer from asbestosis.[120]

3.1.1 Mines

The mining of asbestos in the United States has increased 180 percent in the last 10 years. (See Table 9 in the Appendix.) This mining may constitute a source of air pollution. A high percentage of the increase in domestic production has been credited to California producers, who accounted for 65 percent of the total output in 1966. Four companies produced chrysotile asbestos fiber: Atlas Minerals Corp. and Coalinga Asbestos Co., Fresno County; Pacific Asbestos Corp., Calaveras County; and Union Carbide Corp., San Benito County. The latter company processed the crude material in a plant at King City, Monterey County, whereas the other producers operated plants near the mine sites.[171]

Amphibole asbestos was mined by Powhatan Mining Co. near Burnsville, Yancey County, N.C. Their output increased 66 percent during 1966.[171]

There are four chrysotile mines in Arizona in the Salt River Valley near Globe. Since these mines are underground, only the waste needs to be considered in connection with air pollution (other than the possible pollution from transporting the mineral). Nearly all of the output from these mines was used in the cement industry to manufacture asbestos cement and building products: 28 percent was classified as filter fiber and 2 percent as spinning grade; the rest consisted of sand and waste, floats, or other short fibers. Jacquays Mining Corp. operated the Regal and Chrysotile Mines and shipped the ore to a company mill at Globe after hand-sorting the chrysotile. Western Asbestos Manufacturing Co. operated the Phillips Mine, and the Metal Asbestos Corp. the Lucky Seven Mine.[171]

In Vermont, the Vermont Asbestos Mines Division of the Ruberoid Co. quarried and processed chrysotile near Lowell in Orleans County. Twenty-four grades were produced for spinning, cement stock, paper stock, and other uses. Some waste rock was used for roadstone.[171]

Data on these mines are summarized in Tables 10 and 11 in the Appendix.

Other deposits of asbestos have been found in Georgia and Maryland.

Although no measurements have been made of the asbestos air pollution from mining in the United States, some evidence of the extent of pollution can be drawn from measurements and observations in foreign countries. The extent of air pollution from an asbestos mine in Finland was studied by Laamanen, Noro, and Raunio.[133] They found asbestos dust at distances up to 50 km from the mines, including dust-fall rates ranging from 1.52 g/100 m^2/month at 4 km to 34.6 g/100 m^2/month at 0.5 km. They concluded that asbestos dust is disseminated from mining and milling areas rather extensively and that the degree of pollution varies according to the distance from the mine or mill and the prevailing winds.

Schepers[234] described the dust from asbestos mines and mills in South Africa as dust which "rolled through like a morning mist," producing "itching skins caused by asbestos adhering to our clothes. Even the food at the local hotel was gritty with dust."

Sluis-Cremer[202] reports dust counts in asbestos mines and mills of South Africa as listed in Table 4. He pointed out that living quarters near the mines were polluted with asbestos and that the main source of pollution was airborne asbestos blown off dumps and roads made from the mine tailings.

TABLE 4

DUST COUNTS IN ASBESTOS MINES AND MILLS IN SOUTH AFRICA, 1947[202]

Location	Dust Count, mppcf (mppm^3)	
	Mines	Mill
Northwest Cape Province	2.8-24 (100-840)	10-55 (360-1920)
Transvaal	2.3-6.5 (80-228)	4.6-20 (162-720)

3.2 Production Sources

World production of asbestos during the period 1956 to 1967 increased at the average rate of approximately 13 percent per year. Figure 1 shows that the world production nearly tripled during the period 1945 to 1965, while United States consumption only doubled during the same period. However, during the period 1956 to 1967, domestic apparent consumption fluctuated between 665,000 and 813,000 short tons per year and may be leveling off as substitute materials (such as fiberglass and plastics) provide competition.

The relative importance of the various industrial uses of asbestos is given in Table 12 in the Appendix. It is seen that the highest input of asbestos occurs in the asbestos cement, floor tile, asbestos paper products, and asbestos textile industries. The proportions of asbestos used in various products are shown in Tables 13 and 14 in the Appendix.

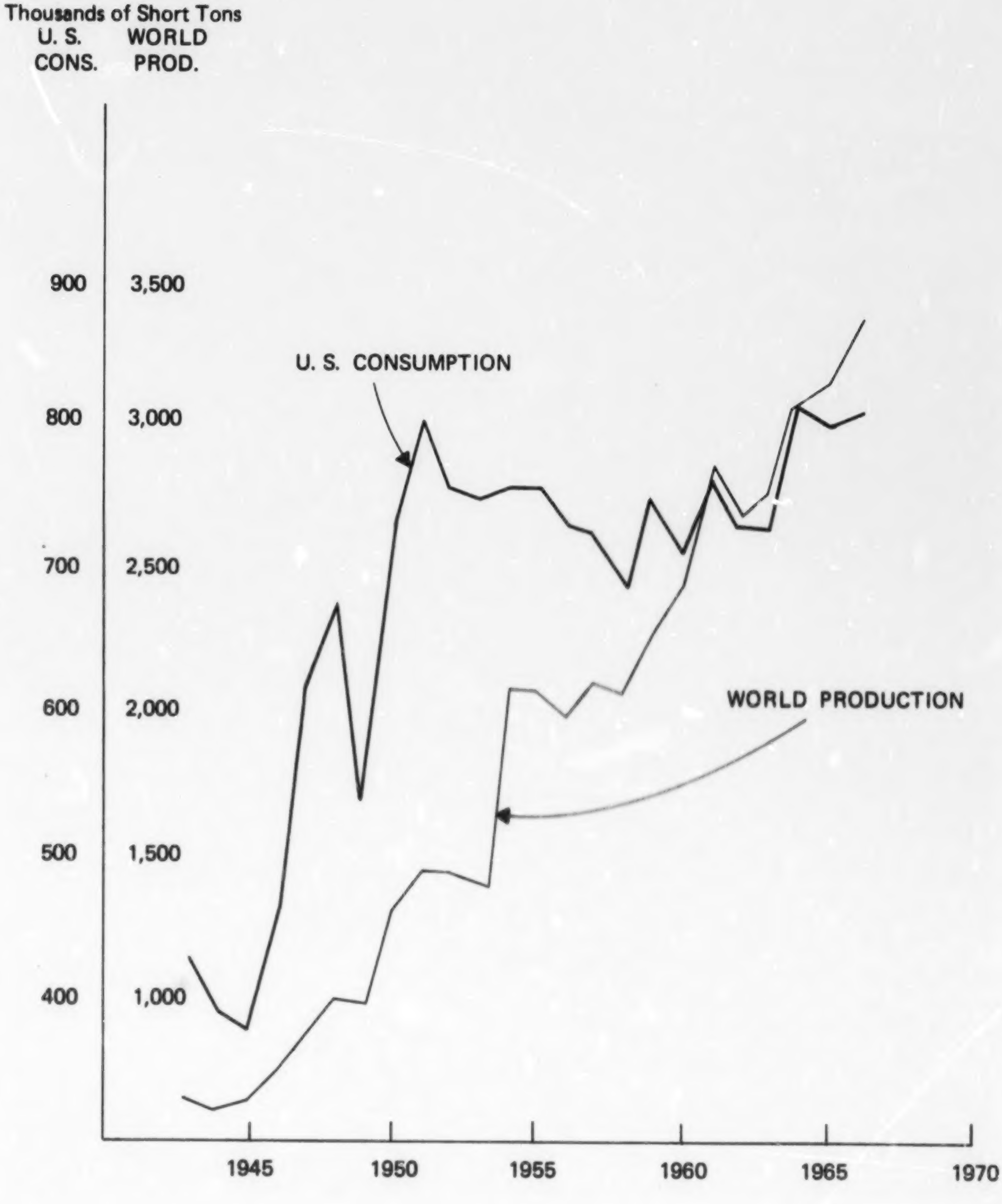

FIGURE 1

Comparison of Trends in World Production and U.S. Consumption of Unmanufactured Asbestos[170,171]

Of the 78,056 short tons of domestic production in 1965, 66 percent were produced in California and 31 percent* in Vermont, amounting to 97 percent of the total production. This 97 percent was produced and processed in five counties.[170] More than 50 percent of the 124 plants comprising the asbestos products industry were located (in 1963) in the States of California, New Jersey, Illinois, Pennsylvania, and Texas, in decreasing order.[41]

Bobyleva _et_ _al_.[24,25] have shown that the air can be polluted by asbestos from plants manufacturing asbestos products. This asbestos may be carried in the air for distances of 25 to 50 miles. In a study of asbestos air pollution from three plants in the U.S.S.R., they found that at a distance of 3 km from the plant, the dust concentration ranged from 0 to 6,000 $\mu g/m^3$; at 1.0-1.5 km it was 3,000-33,000 $\mu g/m^3$; and at 0.5 km it was 6,000 to 34,000 $\mu g/m^3$.

In the United States some attempts[206] have been made to determine the concentration of asbestos near asbestos factories, but the asbestos content of the atmosphere was masked by the other dusts. Asbestos fibers were detected, but a quantitative count was not possible.

3.3 Product Sources

The uses of asbestos are numerous. Some products which use asbestos are the following: asbestos cement which may be applied as mortar or plaster, or sprayed on walls; insulating materials for the covering of pipes, ducts, boilers, cables, and conduits; siding shingles, roofing shingles, tiles, flat and corrugated sheets, wallboard, clapboard, and automobile undercoating; threads, yarns, wicks, cords, tapes, cloths, sheets, and blankets; friction materials, brake linings, clutch facings, gaskets, and lagging cloths; and asphalt tiles, plastics, and similar materials.[108]

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*Extrapolated.

Abrasion of brake linings and clutch facings has been suggested as a primary source of asbestos air pollution. Ayer[13] and Lynch[145] have examined the emissions from brakes on automobiles and found that the fiber is destroyed by the heat of friction. Asbestos crystalline structures are also destroyed and are recognizable only by the chemical composition. Newhouse and Thompson[175] have reported one case of mesothelioma in a mechanic.

The existence of a wide potential for direct or indirect occupational exposure has been cited[32,34,108,120] as a possible explanation for the frequent occurrence of "asbestos bodies" in the general public. Asbestos is now used in more than 3,000 products. Most people working in the construction and demolition of buildings come into contact with asbestos. Electricians and homeowners strip asbestos insulation off wires; the carpenter saws asbestos boards and often pounds the asbestos-insulated furnace ducts to make them fit. As a result, the air around a construction site is contaminated with asbestos fibers, and the foreman, carpenter, painter, plumber, or new occupants all breathe this dust. In most homes, the owner will at some time handle these asbestos products during normal home maintenance. A large number of workers in other industries are similarly exposed. Some of these are listed in Table 15 in the Appendix.

Although only a few of these people work in the asbestos industry, all may have inhaled sufficient asbestos to show "asbestos bodies" upon autopsy. The hazard is there, but how great is the hazard? It will be necessary to obtain quantitative concentration data to delineate its seriousness.

From the above discussion it may be concluded that approximately 100,000 asbestos workers in the United States have a high exposure to asbestos.[195] An additional 3.5 million construction workers—carpenters, welders, electricians, masons, plumbers, steamfitters, tile setters, etc.—are indirectly exposed,either by themselves handling asbestos products or by working on the job with people handling asbestos.[195]

3.4 Environmental Air Concentrations

Only one estimate of the environmental air concentrations of asbestos in the United States was found. Smith and Tabor[206] have roughly estimated that urban air in the vicinity of heavily traveled streets contains 600 to 6,000 particles/m^3. They indicate that the validity of these values is highly suspect because the methods available for the determination of asbestos are inadequate at the concentrations found in the urban air.

4. ABATEMENT

The asbestos industries in the United States have developed elaborate ventilation systems to prevent high dust concentrations which might be inhaled by the workers.[13,95] This dusty air is passed from the ventilators through fabric sleeve filters and then discharged to the atmosphere. The asbestos fibers are easily filtered out since the fibers form a mat which becomes an absolute filter.[13] In addition to the ventilation system, it has been necessary to carry out some operations (such as spinning and weaving) as wet processes to eliminate dust. As a result, the pollution from factories is minimal. Attempts to measure concentrations in the vicinity of an asbestos plant have proved futile with present analytical methods.

Pollution during the transportation of asbestos has been controlled by enclosing the material in plastic-coated bags.

Although the most common procedure used to suppress dust emission is wetting of the material, it is unfortunately not desirable to wet a large number of asbestos products.

In New York, insulators are required to enclose the area when asbestos fireproofing is blown onto steel frames, but even this does not prevent pollution. Asbestos fibers are reported to be a common occurrence in the air around construction sites.[34]

No information has been found on the abatement methods used in United States asbestos mines and in asbestos mills near the mines.

5. ECONOMICS

No information has been found in the literature on the damage costs or economic losses due to the effects of asbestos air pollution on humans, animals, plants, or materials. However, a large fraction of the people in the United States have been exposed to asbestos, including the following:

(1) approximately 100,000 workers using asbestos in their occupations,

(2) approximately 3,500,000 construction workers exposed indirectly to asbestos as they work with asbestos products or near those who handle asbestos products,

(3) approximately 50,000,000 Americans who possibly have "asbestos bodies" in their lungs.

No attempt has been made to assess the cost of health impairment for these people. Workmen's compensation laws for dust diseases are in effect in most States.[234]

No information has been found on the cost of the present and future abatement of air pollution by asbestos in this country. The data in Table 16 in the Appendix, which refer to the asbestos industry in Britain,[95] show that the dust extraction equipment cost alone, for a given size and type of plant, amounts to 27.5 percent of the total capital cost and approximately 7 percent of the operating cost of that plant. The type of control equipment used is primarily designed to meet government specifications relating to occupational health standards.

Data on the production and consumption of asbestos are presented in Section 3.

6. METHODS OF ANALYSIS

Of the methods presently being used[1,2,206] to count dust samples in the asbestos industry,* none is applicable to atmospheric asbestos air pollution. There are at present no proven satisfactory methods for the collection, detection, and identification of asbestos fibers in the 0.1 to 5.0 μ range in ambient air. Satisfactory sampling can probably be accomplished by use of a membrane filter-pump system. The major difficulty lies in the problem of identifying a very few asbestos fibers in the presence of relatively large numbers of a wide variety of other inorganic particulate matter found in the same air. Attempts to determine the asbestos content of urban air have revealed the need for development of new methods. Battelle Memorial Institute is currently developing one such method for the National Air Pollution Control Administration.[205]

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*In all the asbestos monitoring methods used, microscopic counting of the fibrous particles is necessary to determine the proportion of fibrous material, and even then it is not known what fraction of the fibers are asbestos. Counting of fibers by eye under the microscope is tedious and difficult. If the number of fibers is less than 1 percent (<5 wt%) of total dust, the other dust masks the fibers, and quantitative results cannot be obtained.

In parts of the asbestos industry where the asbestos-to-dust ratio is high (>5 wt%), it is often possible to determine the asbestos content indirectly.[136] For example, if the proportion of asbestos in the airborne dust was known by microscopic count for a given sampling location, the concentration (at least the order of magnitude) could then be inferred from a simple measurement of the concentration of the total dust.

Modern analytical methods and instrumentation used in the asbestos industry are listed below:

Microscopic particle counting of samples on membrane filters[1,2,14,15,57,97,136,187]

Thermal precipitators[1,2,97,136,187]

Impingers[1,2,14,15,136,187]

Royco particle counter[1,2,136,187]

Mass concentration methods[1,14,136,187]

Microsieving[116]

Digestion[116]

Column chromatography of organics adsorbed on the surface[116]

X-ray diffraction[14,46,47,116]

Low-temperature ashing[116]

Atomic adsorption spectrophotometry[14,116]

Electron microprobe[116]

Neutron activation[116]

Owens jet counter[1,2]

Konimeter[1,2]

7. SUMMARY AND CONCLUSIONS

Asbestos is an air pollutant which carries with it the potential for a national or worldwide epidemic of lung cancer or mesothelioma of the pleura or peritoneum. Asbestos bodies have been observed in random autopsies of one-fourth to one-half of the population of Pittsburgh, Miami, and San Francisco and will probably be found in the people of every large city. Although asbestos has been shown to produce asbestosis, lung cancer, and mesothelioma in asbestos workers, the relationship between "asbestos bodies" and cancer or asbestosis has not been determined.

The latent period required to develop asbestosis, lung cancer, or mesothelioma is 20 to 40 years, and the exposure required to cause asbestosis has been estimated to be 50 to 60 mppcf-years. No such exposure relationship has been established between asbestos and lung cancer or mesothelioma. Asbestosis, lung cancer, and mesothelioma are all diseases which, once established, progress even after exposure to dust ceases.

Experiments with animals have shown that animals may develop asbestosis or cancer after inhaling asbestos.

No information has been found on the effects of asbestos air pollution on either plants or materials.

The likely sources of air pollution appear to be the vast number of asbestos products used in our modern society, particularly in the building industry. Mines, factories, and shipping yards may also constitute pollution sources.

The world production of asbestos has approximately doubled over the past 10 to 12 years, whereas the domestic consumption has apparently remained relatively constant. These results indicate that while the asbestos air pollution problem of the world may be increasing, the air pollution potential in the United States has remained relatively unchanged. The increase in the number of lung cancer or mesothelioma cases reported in the current literature may be due to increases in asbestos use that occurred 20 to 40 years ago. Moreover, the effects of the asbestos being inhaled today may not be reflected in the general health of the population until the 1990's or the next century.

The number of people exposed to asbestos has been estimated to be 100,000 asbestos workers, 3.5 million people working in areas where asbestos is handled in ways which emit small quantities of dust, and 50 to 100 million people who have breathed or will breathe enough fibers to show positive "asbestos bodies" at autopsy.

No measurements have been made of the concentration of asbestos in urban air. A single estimate of 600 to 6,000 particles per cubic meter has been reported.

Bag filters are used to control asbestos in the exhaust gases from asbestos factories. Wetting the asbestos or its products has also been used to keep the dust from becoming airborne. The cost of abatement equipment in a British factory amounted to 27.5 percent of the total factory cost and approximately 7 percent of the operating cost. Similar data on the costs of abatement or economic losses due to asbestos air pollution in the United States were not found.

No satisfactory method is available to determine the asbestos content at the concentration found in the ambient air. One method is presently under development.

Based on the material presented in this report, further studies are suggested in the following areas:

(1) Identification of the fibers in the lungs of deceased members of the general public should be completed. (Two studies are in progress.)

(2) The relationship between these "asbestos bodies" and lung cancer or mesothelioma should be determined.

(3) The time-concentration relationship between inhalation of asbestos fibers and lung cancer and mesothelioma should be determined.

(4) Methods must be developed to determine the asbestos concentration in air. (One method is under development.)

(5) The concentrations of asbestos fibers in urban air, near mines and factories, and in the vicinity of building construction and demolition should be measured.

(6) The methods and cost of control of asbestos emission from mines, construction or demolition sites, and other normal uses should be determined.

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APPENDIX A

TABLE 5

CANCER OF THE LUNG AMONG ASBESTOS WORKERS[43]

Place	Population Studied	No. of Workers	Years Followed	No. With Lung Cancer	Comparison Groups
United Kingdom	Reported deaths from asbestos	235	1924-1947	31/235 (13.1%)	Silicotics (1.32%)
United Kingdom	Cases of asbestosis in 1,247 autopsies with pneumoconiosis	121		17/121	Silicotics (6.9%)
United Kingdom	Asbestos textile workers, industry areas, 20 years' or more exposure	113	1922-1953	11/39 deaths	0.8 Expected
United Kingdom	Reported deaths from asbestosis	365	1924-1955	65/365 (17.8%)	
Quebec	Chrysotile miners and mill workers with over 5 years' employment	5,958	1950-1955	9/187 deaths	6+ Expected
Pennsylvania	Workers in asbestos products plant employed in 1938-39, aged 25-64	1,495	1940-1960	19/186 deaths	5.61 Expected
New York & New Jersey	Insulating workers, over 20 years' union membership	632	1932-1962	45/255 deaths	6.6 Expected

(continued)

TABLE 5

CANCER OF THE LUNG AMONG ASBESTOS WORKERS (Continued)

Place	Population Studied	No. of Workers	Years Followed	No. With Lung Cancer	Comparison Groups
California	Insulating workers, mixed, 15 years in trade, aged 35-64	529	1954-1957	10/41 deaths	2.8 Expected
Dresden	All asbestos trade mixed exposures	2,636	1924-1963	34/150 deaths	11.4 Expected
United States	Asbestos textile workers employed in 1948-1951, aged 15-64	2,833	1951-1963	24/285 deaths	11.9 Expected
United Kingdom	Reported deaths from asbestosis	584	1924-1963	146/584 (25%)	
Bulgaria	Agricultural workers near an asbestos mine	3,325	1962	155/3325 asbestosis	
New York	Asbestos insulators employed more than 15 years, aged 40-80	152	1945-1965	18/46 deaths	3.1 Expected
Pennsylvania	Asbestos textile and friction workers	68	1957-1962	13/68 deaths	

APPENDIX

TABLE 6

"ASBESTOS BODIES" IN CONSECUTIVE OR RANDOM AUTOPSIES

Location	Total Cases	% Positive	% Positive by Age*							Sex % Positive*		Year of Study	Reference
			<24	25-34	35-44	45-54	55-64	65-74	>75	Female	Male		
Capetown	500	26.4	.5 (4.3)	2.4 (25.6)	4.4 (28.4)	4.6 (22.4)	7.4 (31.6)	7.4 (28.0)		7.8 (20)	18.6 (30.4)	1960	214,216
Miami	500	27.2	0	0	1 (16.1)	3.4 (23)	5.6 (27.2)	9.2 (30.6)	93 (31.9)	8 (20.4)	19.2 (31.6)	1961	214,216
Pittsburgh	100	41	0 (0)	5 (83)	5 (46)	3 (38)	13 (54)	8 (40)	6 (30)	16 (34)	25 (47)	1964	40
Milan	100	51		(←	3 (14)	→)	30 (60)		18 (66)	16 (44)	35 (54)	1966	67
Tyneside	311	20.3	0 (0)	0.3 (25)	1.6 (19.2)	2.9 (17.3)	4.8 (18.5)	7.7 (28.9)	2.9 (15)	4.2 (13)	16.1 (25.5)	1967	11
Jerusalem	100	26	←	1 (14)	→)(←		7→ (21)	←18 (30)	→)	10 (29.1)	16 (22.2)	1967	184
Glasgow	100	23	0	0	0	1 (12)	4 (22)	11 (32)	7 (19)	0 (0)	23 (37)	1967	188
Finland	264	57.6	1.5 (57)	3.0 (57)	3.8 (71)	8.7 (55)	20.0 (64)	16.7 (58.7)	3.8 (34.5)	23.9 (54.3)	33.7 (60)	1966	167
Sweden	34	35.3	0	0	0	0	11.7 (80)	14.7 (25)	18.8 (43)	14.7 (38)	20.6 (33)	1966	82

(continued)

APPENDIX

TABLE 6

"ASBESTOS BODIES" IN CONSECUTIVE OR RANDOM AUTOPSIES (Continued)

Location	Total Cases	% Positive	% Positive by Age*							% Positive*		Year of Study	Reference
			<24	25-34	35-44	45-54	55-64	65-74	>75	Female	Male		
Johannesburg		39.2										1965	234
San Francisco		42										1966	43
Belfast	200	20										1965	60
London	50	6										1964	102
Montreal	100	48		←	3→ (30)	←18 (50)	→	←27 (50)	→	16 (36)	32 (57)	1966	7
New York	355	50.5								3.9 (29.8)	46.5 (53.7)	1966	195

*Numbers in parenthesis represent percentage of group-age or sex.

APPENDIX

TABLE 7

COMPOSITION AND PROPERTIES OF ASBESTOS MINERALS[66]

	Chrysotile	Crocidolite	Amosite
Approximate Formula	$3MgO-2SiO_2 \cdot 2H_2O$	$Na_2O-3FeOFe_2O-8SiO_2 \cdot H_2O$	$1.5\ MgO-5.5FeO-8SiO_2 \cdot H_2O$
Percentage of Major Components			
Silica SiO_2	40.3	51.4	49.3
Alumina Al_2O_3	0.7		
Ferrous Oxide FeO	1.0	20.3	40.9
Ferric Oxide Fe_2O_3	1.5	17.5	0.4
Manganous Oxide MnO		0.1	0.7
Calcium Oxide CaO	0.2	0.8	0.4
Magnesium Oxide MgO	42.4	1.4	5.7
Sodium Oxide Na_2O		6.2	0.2
Potassium Oxide K_2O			0.3
Carbon Dioxide CO_2	0.2	0.4	0.2
Water of Crystallization H_2O	13.7	1.9	1.9

(continued)

TABLE 7

COMPOSITION AND PROPERTIES OF ASBESTOS MINERALS (Continued)

Approximate Formula	Chrysotile $3MgO-2SiO_2\cdot 2H_2O$	Crocidolite $Na_2O-3FeOFe_2O-8SiO_2\cdot H_2O$	Amosite $1.5\ MgO-5.5FeO-8SiO_2\cdot H_2O$
Trace Organic Impurities			
Oil-wax (mg/100 g fiber)	4-7.6	4-200	4-20
Benzo(a)pyrene (μg/100 g fiber)	none detected	0.2-24	0.2-2.4
Trace Inorganic Impurities (μg/g fiber)			
Pb	2	5	20
Sn	<5	<5	<5
Ga	<2	<2	2
Bi	<5	<5	<5
V	50	<2	<2
Mo	<2	<2	<2
Cu	35	7	7
Ti	50	50	300
Ag	<0.2	0.2	0.2
Ni	5,000 (1,000-14,000)	<10 (<100)	1,000 (<100)
Zv	<200	700	1,000
Co	<5 (<100)	<5 (<100)	<5 (<100)
Mn	130 (400-500)	180 (200)	7,000 (7,900)
Cr	1,000 (400-900)	20 (<100)	150 (<100)

(continued)

APPENDIX

TABLE 7

COMPOSITION AND PROPERTIES OF ASBESTOS MINERALS (Continued)

Approximate Formula	Chrysotile	Crocidolite	Amosite
Radioactive Contaminants ($\mu\mu$c/g fiber)			
K^{40}	0.14	0.02	0.55
Th^{238}		<0.01	0.05
Ra^{226}	0.07		0.15
Physical Properties			
Flexibility	Very flexible	Fair to good	Good
Length	Short to 3"	Short to 3"	¼" to 6"
Texture	Harsh to silky	Harsh to soft	Coarse but pliable
Tensile strength	Very high	Very high	Fair
Acid resistance	Fairly soluble	Very good	Good
Heat resistance	Good	Poor	Good
Spinnability	Very good	Fair	Fair

APPENDIX

TABLE 8

WORLD PRODUCTION OF ASBESTOS[170]
(Short Tons)

Location	1962	1963	1964	1965	1966[b,e]
North America					
Canada (sales)	1,215,814	1,275,530	1,420,769	1,387,555	1,479,281
United States (shipments)	53,190	66,396	101,092	118,275	125,928
South America					
Argentina	203	365	542	243[c]	240[a]
Bolivia (exports) . .	56	10	7	3	4
Brazil	4,900[a]	1,440[f]	1,430[a,c,f]	1,204	1,820
Europe					
Austria	503	638			
Bulgaria	1,323[c]	1,323[c]	1,433[c]	1,433[c]	1,430[a]
Finland[g]	10,869	10,201	11,611	13,307	13,250
France	28,034[c]	26,094[c]	24,289[c]	7,506[c]	7,720[a]
Greece		74	65[a]	85[a]	85[a]
Italy	60,860	63,016	75,573	79,214	90,464
Portugal		29		53[c]	10
U.S.S.R.	710,000[a,c]	755,000[a,c]	810,000[a,c]	865,000	925,000[a]
Yugoslavia	7,401	9,074	9,280	10,585	8,411
Africa					
Botswana	2,375	2,368	2,161	888	880[a]
Kenya	212	78	204	136	73
Mozambique	370				
Rhodesia, Southern . .	142,195[c]	142,254[c]	153,450	176,149[c]	175,000[a]
South Africa	221,302	205,744	215,592	240,752	276,597
Swaziland	32,830	33,350	39,862	40,884	36,142
United Arab Republic .	606	192	1,739	3,225	2,057

(continued)

TABLE 8

WORLD PRODUCTION OF ASBESTOS (Continued)
(Short Tons)

Location	1962	1963	1964	1965	1966[b,e]
Asia					
China	100,000	110,000	130,000	140,000	140,000
Cyprus	22,391	19,962	13,755	17,622[c]	24,449
India	1,865	3,038[c]	3,710[c]	4,989	7,646
Japan	15,407	18,210	17,979	16,451[c]	17,067
Korea, South	1,333	2,120	1,402	1,710[c]	687
Philippines	1,037	421	586		
Taiwan	525	604	526	883	721
Turkey	709	408	1,291	1,376	1,258
Oceania					
Australia	18,416	13,374	13,545	11,647	13,472
New Zealand	457	439			
World Total[a] . . .	2,655,000[c]	2,760,000[c]	3,050,000[c]	3,140,000[c]	3,350,000

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[a] Estimated.

[b] Preliminary.

[c] Revised.

[d] Asbestos also is produced in Czechoslovakia, Eritrea, Malagasy, North Korea, and Rumania. No estimates for these countries are included in the total because production is believed to be negligible.

[e] Compiled from data available May 1967.

[f] Bahia only.

[g] Includes asbestos flour.

TABLE 9

THE PRODUCTION AND APPARENT CONSUMPTION OF ASBESTOS IN THE UNITED STATES[41,170,171]

	Quantities in Short Tons			
Year	Production	Imports	Exports	Consumption
1967	123,190	645,110	47,710	720,580
1966	125,928	726,459	46,996	805,391
1965	118,275	719,559	43,126	794,708
1964	101,092	739,361	27,147	813,306
1963	66,396	667,860	10,044	724,212
1962	53,190	675,953	2,949	726,194
1961	52,814	616,529	3,799	(665,440)
1960	45,223	669,945	5,525	709,193
1959	45,459	713,047	4,461	754,045
1958	43,979	644,331	3,026	685,284
1957	43,653	682,732	2,893	723,492
1956	41,312	689,910	2,950	782,272
1955	50,431	716,480	7,001	759,910
1950				727,002
1945				378,030
1940				270,000
1935				170,000

	Values (X 1,000)			
			Exports	
Year	Production	Imports	Asbestos	Asbestos Products
1967	$11,100	$66,000	$6,030	
1966	11,056	73,100	5,763	$21,963
1965	10,162	70,457	5,294	19,139
1964	8,143	72,973	3,199	16,288
1963	5,108	61,739	1,304	16,267
1962	4,677	64,112	598	14,274
1961	4,347	(63,000)	759	
1960	4,231	63,345	857	
1959	4,391	65,006	793	
1958	5,127	58,314	424	13,233
1957	4,918	60,104	350	15,223
1956	4,742	61,939	375	14,181
	4,534	59,339	1,497	12,464

APPENDIX

TABLE 10

REGIONAL DISTRIBUTION OF ASBESTOS MINING AND PROCESSING[170,171]
(Ranking and Production)

Rank	State			Quantity (Short Tons)			Value (X 1,000)		
	1960	1964	1966	1964	1965	1966	1964	1965	1966
1	Vt.	Calif.	Calif.	55,041	74,587	81,671	$4,419	$6,177	$6,945
2	Ariz.	Vt.	Vt.	*	*	*	*	*	*
3	N.C.	Ariz.	Ariz.	*	3,469	*	*	441	*
4	Calif.	N.C.	N.C.	*	*	*	*	*	*
				(55,041)	(78,056)	(81,671)	(4,419)	(6,177)	(6,945)

*Data withheld to avoid disclosure of producer's confidential information to the Bureau of Mines.

APPENDIX

TABLE 11

ASBESTOS MINES IN THE UNITED STATES, 1966[170]

State	Mineral	Production Rank[a]	Name of Producer	Location--County or City of Mine	Location--County or City of Processing Plant
California	Chrysotile	1	Atlas Minerals Corp.	Fresno County	Near Mine
	Chrysotile	2	Coalinga Asbestos Co.	Fresno County	Near Mine
	Chrysotile	3	Pacific Asbestos Corp.	Calaveras County	Near Mine
	Chrysotile	4	Union Carbide Corp.	San Benito County	Monterey County (King City)
			Ash Bonding Co.[b]	Napa	
Vermont	Chrysotile	1	Vermont Asbestos Mines Div. of Ruberoid Co.	Orleans County (Lowell)	
Arizona	Chrysotile	1	Jacquays Mining Corp.	Salt River Valley (North of Globe)	
	Chrysotile	2	Western Asbestos Mfg. Co.		
	Chrysotile	3	Metate Asbestos Corp.		
			Kyle Asbestos Mines of Ariz.[a]		
			LeTourneau Asbestos Corp.[a]		
North Carolina	Crocidolite	1	Powhatan Mining Co.	Yancey County (Burnsville)	

[a] Ranked by production only within the State.

[b] Not in operation during previous years.

APPENDIX

TABLE 12

APPARENT ASBESTOS CONSUMPTION, 1965[170]
(In Thousands of Short Tons)

Use	World[97]	United States*
Asbestos Industry Production Mining and Processing		78
Asbestos in Products		
Textiles	66	17
Cement	2,190	548
Friction Materials	111	28
Asbestos Paper	220	55
Floor Tile	307	77
Paints, Roof Coating, Caulks	85	21
Plastics	21	5
Miscellaneous	221	55
Total Products	3,221	806

*Based on 25% of world consumption.

APPENDIX

TABLE 13

PROPORTION OF ASBESTOS IN VARIOUS ASBESTOS PRODUCTS[170]

Product	Percent Asbestos[a]
Asbestos textiles	80-100
Asbestos cement	15-90[b]
Friction materials and gaskets	30-80
Asbestos paper and products	80-90
Floor tile	10-30
Other asbestos products	U.D.[c]

[a] Chrysotile asbestos is used unless otherwise stated.

[b] 15% to 90% chrysotile, with some materials containing as much as 85% amosite and small amounts of crocidolite and anthophyllite.

[c] These products contain undetermined quantities of chrysotile, tremolite, actinolite, and anthophyllite.

TABLE 14

QUANTITY AND VALUE OF ASBESTOS INPUT BY INDUSTRY 1963[170]

Product	Quantity		Value	
	Short Tons(000)	Fraction of Total	$(000,000)	Fraction of Total
Asbestos textiles	66	0.02	26.4	0.06
Asbestos cement	2,190	0.68	328.5	0.78
Friction materials and gaskets	111	0.04	11.1	0.03
Paper and products	220	0.07	19.8	0.05
Floor tile	307	0.09	13.2	0.03
Paint and coating fillers	85	0.02	3.7	0.01
Plastics	21	0.01	9.2	
Other	220.7	0.07	19.7	0.04
Total	3,220.7	1.00	423.3	1.00

APPENDIX

TABLE 15

POPULATION GROUPS WITH OCCUPATIONAL AND ENVIRONMENTAL EXPOSURE TO ASBESTOS[108]

Occupational Groups	Nonoccupational Groups
Asbestos rock miners, loaders, truckers, crushers, millers, asbestos spinners, weavers, electrical appliance and wire manufacturers, masons, carpenters, heating equipment workers, rubber workers, shingle and tile manufacturers, building material manufacturers, filtering material manufacturers, molders of asbestos products, asbestos-asphalt makers, putty manufacturers, asbestos cement makers, asbestos paper, cardboard and brake-lining producers, asbestos felt insulation workers, asbestos sound insulation workers, asbestos insulators, pipe coverers, asbestos tube wrappers, asbestos cork insulation workers, construction workers, automobile makers, garage attendants	Residents in vicinity of asbestos processing and textile mills inhaling plant effluents polluted with asbestos dust, and individuals living and working along roads on which asbestos is trucked; residents in the vicinity of asbestos mines; residents in vicinity of building construction and demolition, inhabitants of homes or offices with asbestos acoustical tile

TABLE 16

ASBESTOS CONTROL EQUIPMENT[95]

Cost Data	
Asbestos Textile Industry[a]	
Dust extraction equipment	
Total capital cost (fixed)	~ $1,500,000
Operating cost per year	~ $ 250,000
Operating cost/labor cost	~ 7. %
Operating cost/total cost conversion	~ 2.7%
Asbestos Mines[b]	
Dust extraction equipment	
Total capital cost (TCC)	~ $ 360,000
(TCC/TCC of plant) x 100	~ 27.5%
Operating cost (per year)	~ $ 195,000

Specifications	
Asbestos Textile Industry[a]	
Volume of dust-containing air extracted from textile machines	1,000,000 ft^3/min or 700 ft^3/min/operative
Quantity of asbestos dust filtered per year (at above rate)	700 tons or 2.8 tons/working day
Asbestos Mines	
Total installed horsepower	796
Part used to generate air for dust removal	230 (29%)
Total air needed (for aspiration and dust removal) per pound of fiber produced	1,350 ft^3

[a] Figures apply to the Turner Bros. Asbestos Co. plant at Rochdale, England.

[b] Figures apply to the Cape Asbestos Co. at Penge in the Transvaal. The most modern mill (in Canada), which is 10 times larger, needs only about half the quantity of air stated (using gravity instead of air-swept mills and horizontal transportation of ore).

APPENDIX

TABLE 17

ANALYSIS OF ASBESTOS AND ASBESTOS PRODUCTS EXPORTS AND IMPORTS[170]

	Exports				Re-exports			
	Short Tons		Dollars (000)		Short Tons		Dollars(000)	
	1965	1966	1965	1966	1965	1966	1965	1966
Crude and spinning fibers	1,251	1,455	326	325	50	176	10	30
Nonspinning fibers	24,221	28,017	3,622	3,973	81	130	13	21
Waste and refuse	17,523	17,218	1,323	1,414				
Total	42,995	46,690	5,271	5,712	131	306	23	51
Gaskets and packing	1,732	2,678	4,528	5,261			1	1
Brake lining	3,065	3,630	4,728	5,236	1	1	2	2
Clutch lining (number)	2,020,864	2,246,986	1,691	1,897	5,000	5,000	4	1
Textiles and yarn	794	900	1,067	1,326				
Shingles and clapboard	5,465	10,010	1,096	1,797	113	231	37	41
Asbestos-cement	6,101	4,742	1,588	1,332				
Sub total			14,698	16,849	5,114	5,232	44	45
Other products	*	*	4,389	5,058	*	*	8	11
Total			19,087	21,907			52	56

	Imports			
	Short Tons		Dollars (000)	
	1965	1966	1965	1966
Chrysolite				
Crude	12,496	6,596		
Spinning/ Textiles	17,339	16,839	6,245	6,319
All Other	643,149	642,894	55,077	56,308
Total	672,984	666,329		
Crocidolite	21,165	26,995		
Amosite	17,042	23,934		
Total	711,191	716,258	70,454	73,100

*Not available.

APPENDIX

TABLE 18

SELECTED STATISTICS FOR THE ASBESTOS MANUFACTURING INDUSTRY[41]
(Employment Size)

Number of Employees*	Number of Companies	Number of Plants	Number of Production Workers	Value of Shipments $ (000)
1-49	39	39	308	8,264
50-99	6	6	231	4,827
100-2,499	10	17	2,445	89,131
over 2,500	18	62	12,754	407,014
Total	73	124	15,738	509,236

*The employment size class is determined by the total company employment in all manufacturing activities in the U.S., including central offices and auxiliaries serving manufacturing establishments. All establishments of a company are therefore included in the same employments size column regardless of establishment size.

APPENDIX

TABLE 19

SELECTED STATISTICS FOR THE ASBESTOS PRODUCTS INDUSTRY[41]

Expenditures (in $000)	1958	1963
New plant and equipment		
New structures	2,419	2,613
New machinery and equipment	10,418	9,768
Total	12,837	12,381
Used plant and equipment	428	1,289
Total	13,265	13,670

TABLE 20

ASBESTOS USES[85]

Textiles:

Varieties used: Chrysotile, crocidolite, and in part amosite

Yarns and Cords:

Processes: Weaving of yarns and cords
Braiding (interlacing)

Classification of chrysotile fabrics:

Class	Quality Code	Asbestos Content (%)
1	AAAA	75-79.9
2	AAA	80-84.9
3	AA	85-89.9
4	A	90-94.9
5	Underwriters Commercial	95-100

Sealing and Packing Materials:

Packing (woven fabrics)
stuffing for boxes and sleeves
manhole rings, boiler covers

Flat Packing:

Gaskets, flanges (on pipes) and containers

1. Without metal: high pressure gasket sheets (rubber)
2. With metal: material for sealing cylinder heads and exhausts in motors and combustion engines, and for sealing compressors and turbines

Asbestos Boards and Papers:

Boards
- Filtering and clarifying
- Coverings, coatings, casings, and jacketings for all kinds of surfaces
- Manufacturing of welders' and melters' shields
- Slideways in the glass industry
- Handles and fire-doors
- Auto Parts
- Safes
- Protective walls
- Curtains, etc.

(continued)

TABLE 20 (Continued)

ASBESTOS USES[85]

Sheets

Inner/outer linings of furnaces and heating vessels, drying ovens, incubators, heaters, climate-controlled spaces, etc.

Plates

Insulating buildings against vibrations (aluminum-asbestos)
Solar-heat reflecting surfaces (70% of solar heat)

Special Asbestos Papers

Filters

Asbestos Cement (10 to 25% asbestos):

Slabs
Corrugated sheets
Pipes
Corrugated tiles for roofs in industry, agriculture, and dwellings
Planks for platforms in buildings under construction
Balcony canopies
Rain gutters
Interior walls
Ventilating shafts
Air conditioning assemblies
Pressure piping (for underground drinking water distribution systems, fuel gas, and sewage)
Cooling towers (electricity-generating stations)

Thermal Insulants and Fire-Proofing:

Sprayed asbestos (**insulant** in both heating and refrigeration), **sound absorbent** (eliminates booming and improves acoustical properties of walls and ceilings)
Magnesia asbestos (85% magnesia, 15% asbestos) as thermal insulant for covering pipes

Friction Material:

Woven: Brake lining
Nonwoven: Clutch lining
Transmission lining

(continued)

TABLE 20 (Continued)

ASBESTOS USES[85]

Asbestos Plastics:

- Flooring tiles (asbestos-asphalt tiles and, increasingly, asbestos-polymers of vinyl)
- Pressed or molded (thermal insulation and in electrical machinery)
- Resinated asbestos felt (manufacturing of wings and firing of missiles and expansion cones for nozzles of boost motors). Other uses in aircraft industry: nozzles for motor tubes, missile tailpipes, and missile-heat barriers; fuselages for guided missiles, fuel tanks for fighter bombers, cabin floors, etc.
- Radar (large molded reflectors and scanners)

Asbestos Acid-Resistant Compositions:

- Used mostly in chemical industry

APPENDIX

TABLE 21

1967 LIST OF MANUFACTURED ASBESTOS PRODUCTS[85]

Industry and Product Description	Quantity Measure
Miscellaneous Nonmetallic Mineral Products	
Asbestos Products	
Asbestos Friction Materials	
Brake Linings	
Woven, containing asbestos yarn, tape, or cloth	Linear feet
Molded, including all nonwoven types	Cubic feet
Clutch facing	
Woven, containing asbestos yarn, tape, or cloth	Thousand pieces
Molded, including all nonwoven types	Thousand pieces
Asbestos-Cement Shingles and Clapboard	
Siding shingles and clapboard, including accessories	Squares
Roofing shingles	Squares
Asphalt Floor Tile	Thousand square yards
Asphalt floor tile	
Vinyl Asbestos Floor Tile	Thousand square yards
Vinyl asbestos floor tile	
Asbestos Textiles and Other Asbestos-Cement Products	
Asbestos textiles	
Yarn, cord, and thread	Pounds
Cloth	Pounds
Other asbestos textiles, including roving, lap, wick, rope, tape, carded fibers, etc.	Pounds
Asbestos-cement products	
Flat sheets and wallboard, all thicknesses converted to ¼" basis	100 square feet
Corrugated sheets	100 square feet
Pipe, conduits, and ducts, including pressure pipe	Short tons

(continued)

TABLE 21 (Continued)

1967 LIST OF MANUFACTURED ASBESTOS PRODUCTS[85]

Industry and Product Description	Quantity Measure
Asbestos felts	
Roofing-asphalt or tar saturated	Short Tons
Other	Short Tons
Other asbestos and asbestos-cement products, including millboard and prefabricated housing components	
Gaskets and Insulation	
Gaskets, All Types	
Gaskets (for sealing nonmoving parts)	
Asbestos, asbestos-metallic, and asbestos-rubber	
Packing (except leather, rubber, and metal) and Asbestos Insulations	
Asbestos compressed sheet	Pounds
Packing (for sealing moving parts)	
Asbestos, asbestos-metallic, and asbestos-rubber	Thousand pounds
Insulation materials containing asbestos pipe insulation	
Cellular and laminated	Linear feet
85 percent magnesia	Linear feet
Diatomaceous silica, calcium, silicate, expanded silica, and asbestos fiber	Linear feet
Other pipe insulation	Linear feet
Block insulation, including sheet and lagging	
85 percent magnesia	Thousand board feet
Diatomaceous silica, calcium silicate, expanded silica, and asbestos fiber	Thousand board feet
Other block insulation, including celluar and laminated	Thousand board feet
All other asbestos insulation	

TABLE 22

ASBESTOS PRODUCT MANUFACTURING PLANTS, 1963[41]

Location	Total Plants	No. of Plants with Employment of						
		1-19	20-49	50-99	100-249	250-499	500-999	1,000 or more
New Hampshire								
Belknap	1				1			
Hillsborough	1					1		
Total	2				1	1		
Massachusetts								
Essex	1			1				
Franklin	1	1						
Middlesex	2	1		1				
Suffolk	2	2						
Worcester	1			1				
Total	7	4		3				
Connecticut								
Fairfield	3		1	1				1
Hartford	1	1						
Middlesex	1				1			
Total	5	1	1	1	1			1
New York								
Albany	1						1	
Kings	3	1	1			1		
Orange	1					1		
Suffolk	1	1						
Total	6	2	1			2	1	
New Jersey								
Bergen	1		1					
Essex	3	2	1					
Hudson	1					1		
Mercer	2					2		
Morris	1				1			
Passaic	3	2				1		
Somerset	4				2	1	1	
Union	1	1						
Total	16	5	2		3	5	1	

(continued)

TABLE 22 (Continued)

ASBESTOS PRODUCT MANUFACTURING PLANTS, 1963[41]

Location	Total Plants	No. of Plants with Employment of						
		1-19	20-49	50-99	100-249	250-499	500-999	1,000 or more
Pennsylvania								
Elk	1				1			
Lancaster	1							1
Montgomery	2				2			
Northampton	1			1				
Philadelphia City	3	2				1		
Potter	1	1						
Total	9	3		1	3	1		1
Ohio								
Cuyahoga	1	1						
Paulding	1				1			
Portage	1				1			
Ross	1				1			
Total	4	1			3			
Indiana								
Henry	1					1		
Huntington	1					1		
Kosciusko	1				1			
Lagrange	1	1						
Lake	1				1			
Rush	1		1					
Total	6	1	1		2	2		
Illinois								
Cook	8	5	1			2		
Kankakee	1					1		
Lake	4				2	1	1	
Will	1				1			
Total	14	5	1		3	4	1	
Michigan								
Wayne	1	1						
Total	1	1						

(continued)

TABLE 22 (Continued)

ASBESTOS PRODUCT MANUFACTURING PLANTS, 1963[41]

Location	Total Plants	No. of Plants with Employment of						
		1-19	20-49	50-99	100-249	250-499	500-999	1,000 or more
Wisconsin								
Milwaukee	1	1						
Total	1	1						
Missouri								
St. Louis	2			2				
St. Louis City	3	1			2			
Total	5	1		2	2			
Kansas								
Barton	1	1						
Total	1	1						
Virginia								
Essex	1	1						
Frederick	1					1		
Norfolk City	1	1						
Total	3	2				1		
North Carolina								
Mecklenburg	2			1		1		
Union	1				1			
Total	3			1	1	1		
South Carolina								
Charleston	1					1		
Marlboro	1			1				
Total	2			1		1		
Georgia								
DeKalb	1	1						
Talbot	1	1						
Troup	1				1			
Total	3	2			1			

(continued)

TABLE 22 (Continued)

ASBESTOS PRODUCT MANUFACTURING PLANTS, 1963[41]

Location	Total Plants	No. of Plants with Employment of						
		1-19	20-49	50-99	100-249	250-499	500-999	1,000 or more
Florida								
Dade	1	1						
Total	1	1						
Alabama								
Mobile	1			1				
Total	1			1				
Mississippi								
Hinds	1				1			
Union	1	1						
Total	2	1			1			
Louisiana								
Jefferson	3				2	1		
Orleans	3		1		1	1		
Total	6		1		3	2		
Texas								
Dallas	2	2						
Ector	1	1						
Grayson	1				1			
Harris	3				2	1		
Hill	1				1			
Total	8	3			4	1		
California								
Alameda	2		1	1				
Contra Costa	1			1				
Los Angeles	9	1	2	1	4	1		
Orange	1	1						
Sacramento	1	1						
San Benito	1	1						
San Joaquin	1				1			
San Mateo	1	1						
Santa Clara	1				1			
Total	18	5	3	3	6	1		
UNITED STATES TOTAL	124	40	10	13	34	21	4	2

APPENDIX B

RESPIRABLE ASBESTOS FIBERS

A number of questions arise regarding respirable asbestos fibers. What length of fiber is respirable? What is the particle-to-mass ratio? Should all fibers, whatever their length or diameter, be counted? If not, can any instrument be designed to select the right size distribution in the atmosphere?

Timbrell[218] has studied the deposition of fibrous material in the respiratory system. Fibers 50 or even 200 μ long are found in the lungs because the free-falling speed depends largely on the diameter. Thus, particles less than 3.5 μ (most asbestos particles are less than 0.5 μ) in diameter can possibly penetrate deeply into the lung. The more symmetrical a fiber is, the greater its chance of penetrating. The largest compact particles normally found in the lung are about 10 μ in diameter. Limitation on the lengths of the fibers which reach pulmonary air spaces is imposed by the nasal hairs and the small diameters of the respiratory bronchioles. These limitations are summarized in Table 23.

Respirable fibers have been defined by the British Occupational Hygiene Society[204] as fibers less than 200 μ long, less than 3.5 μ in diameter, and having a length-to-breadth ratio of 3:1. Only the fibers longer than 5 μ in length are counted.

TABLE 23

PENETRATION OF FIBERS THROUGH NASAL HAIRS[218]

Length of Fiber (microns)	% Penetration through Nasal Hairs		
	1st Stage	2nd Stage	3rd Stage
0.5	100	100	100
50	75	57	42
100	53	24	11
150	31	10	3
200	26	5	1
250	20	3	
300	17	2	
350	14	1	

Walter[229] has investigated the mass of average particles in the asbestos textile industry (see Table 24). He found that respirable dust contains approximately 50 percent asbestos and that 10 particles of dust per μg contain 5 x 10^5 particles of asbestos per μg. From this conversion factor the threshold limit value for asbestos can be calculated* as approximately 350 μg/m^3 (5 mppcf).

TABLE 24

PARTICLE-MASS RELATIONSHIP OF ASBESTOS AS A FUNCTION OF FIBER LENGTH

Total Concentration		Incineration Residue μg/m^3 (approx)	Fiber Length in Microns (approx)
Particles*/cm^3	μg/m^3 (approx)		
100	100	50-60	< 150
200	400	200-300	< 500
400	1,000	700-800	< 700
600	2,500	1,800-2,000	<10,000

*Particles counted with a konimeter.

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*5 mppcf=177 x 10^6 p/m^3=350 μg/m^3. The concentration estimated in air is 600-6,000 p/m^3=1.2 - 12 x 10^{-9} μg/m^3.

Finally, there remains the problem of counting respirable fibers in ambient air. It appears that a fairly sophisticated instrument will be required which can (1) separate the other particles from fibers, (2) identify the asbestos fibers in a host of other fibers, and (3) count only those fibers longer than 5 μ and shorter than 200 μ with diameters less than 3.5 μ.